Table 6. Relation between indoor CO, CO personal exposure and COHb levels.

	Pupils from homes using coal as fuel	Pupils from homes using gas as fuel
Indoor CO (mg/m³)	9.11	4.38
CO exposure (mg/m³)	7.90	4.14
pupils' COHb (%)	1.11	0.50

Pulmonary functions

Although the indoor air pollution in winter was serious, the pupils pulmonary functions did not seem to be affected by the pollution.

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REFERENCES

1. Chinese Academy of Preventive Medicine, Measurement methods of atmospheric pollution, Chinese Chemical Industrial publication Company. The first edition, Beijing 1984.
2. X.Y. Guo, G.H. Jians, A sampling investigation of the daily consumption of time of the people in Wuhan City, Environment and Health, Vol.2, No.3, 1985.(Chinese Journal).
3. Yao Zhiqi, Environmental Hygiene (the second edition), Chinese people Health publishing House, 1987, Beijing.

EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE AND FEMALE LUNG CANCER IN GUANGZHOU, CHINA

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ABSTRACT

Cigarette smoking is widely accepted as a major risk for human lung cancer. However, the relationship between ETS exposure and female lung cancer is being debated. Since 1980 to 1988, there have been 5,546 cases (M: 3,760; F: 1,786) of deaths from lung cancer in Guangzhou, and 811 cases (M: 209; F: 602) of them were never smokers. In this group, 552 cases (M: 94; F: 458) were from ETS exposure. In order to ascertain the relationship between ETS exposure and lung cancer, some epidemiological analyses have been performed as follows: (1) Comparisons of medical histories between ETS and Non-ETS exposure of never smokers. (2) Conditional logistic regression analyses of never smokers. (3) A casecontrol study of female never smokers. (4) ETS exposure and cell type of lung cancer. All results of these studies demonstrated that exposure to ETS had no association with female lung cancer.

INTRODUCTION

Cigarette smoking is widely accepted as a major risk for lung cancer in both males and females. However the relationship between exposure to environmental tobacco smoke (ETS) and female lung cancer is a subject of considerable controversy. Because of a long latency required for lung cancer to be induced, and since ETS exposure is multifaceted, in order to certify the relationship between ETS exposure and female lung cancer, at least, two condition should be met in studying the effects of ETS. First, the subjects must be truly and solely exposed to ETS. Second, the results of epidemiological study can be elucidated the mechanisms for the pathogenesis of lung cancer, especially in the relationship between inducing factors and lung cancer cell type .

MATERIALS AND METHODS

Case History

Guangzhou covers an area of 50 square kilometers, and about 2 million people live there. It is divided into four districts -- LW, YX, DS and HZ, and contains 63 local police stations. Beginning in 1980 to 1988, every case of lung cancer death was further analyzed using a standardized questionnaire containing 31 questions. Information was obtained retrospectively from relatives and verified by the hospital records. The questionnaires were administered by trained medical personnel and data entered into a computer. Since in China all deaths, including time and cause, had to be reported to the local police station,

the generated data were considered to be accurate. Furthermore, if lung cancer deaths had been of ETS exposure,the respondents, relatives of the dead, mainly the active smokers, in this situation, who responded, gave a highly reliable smoking history and proximity of the ETS exposure.

Comparison of medical history between ETS and Non-ETS exposure of never smokers

The 811 cases of lung cancer deaths of never smokers were further grouped as follows:

	Male	Female	Total
Group 1 ETS exposed	115	144	259
Group 2 Non-ETS exposed	94	458	552

In these never smoking groups, 794 cases had chronic bronchitis or emphysema record (positive and negative), and 465 cases had lung cancer metastasis record. The effects of ETS on such medical histories have been compared.

Conditional logistic analyses of never smokers

In 1985, there were 806 cases of deaths from lung cancer, 120 of them were never smokers. A Conditional logistic analysis was performed on those who never smoked (M:28;F:92). Matched with two control groups, one a non-respiratory system disease, another one a non- respiratory cancer. All control cases were of same sex, age (±2 years), residence and having never smoking. Investigation items included: x_1- history of respiratory disease; x_2- consumption of fresh vegetables; x_3- history of contact with toxic substances prior to death; x_4- ETS exposure; x_5- indoor air pollution; x_6- size of living area; x_7- situation of kitchen; x_8- cooking fuel; x_9- participation in cooking; x_{10}- family history of cancer.

A case-control study on non-smoking females

In 1986, there were 236 females who died from lung cancer, 75 of them had never smoked, and the ETS exposure was limited to a husband. A casecontrol study was performed on these cases using two control groups. One of non-tumor diseases (128 cases), another one of tumors other than lung cancer (126 cases). All control cases were of same sex, age (±2 years), residence and having never smoked.

ETS exposure and cell type

It is generally known that the cell type of lung cancer induced by smoking is mainly an epidermoid carcinoma and not adenocarcinoma. Consequently, it is reasonable to believe that if passive smoking can cause lung cancer, the cell type must be epidermoid carcinoma and not adenocarcinoma. In this study, the constituent ratio of cell type of 192 never smoking lung cancer deaths (M: 53, F: 139) was compared between ETS and Non-ETS exposure.

RESULTS

Comparison of medical histories between ETS and Non-ETS exposure of never smokers

The influence of ETS on the occurrence of respiratory illness (chronic bronchitis, emphysema and lung cancer metastasis) is shown in table 1. No effect of exposure to ETS was found.

Table 1. Relationship between ETS exposure and some medical history in never smoker lung cancer deaths.

		Number of Family Smoker											
		1		2		3		Total		Non-ETS exposure		P-Value
Medical history		No.	%	No.	%	No.	%	No.	%	No.	%	
Chron. bronchitis												
Male	Yes	6	11.5	7	25.0	1	8.3	14	15.2	15	13.5	
	No	46	88.5	21	75.0	11	91.7	78	84.8	96	86.5	P>0.05
Female	Yes	28	11.1	22	17.6	12	16.0	62	13.7	24	17.4	
	No	225	88.9	103	82.4	63	84.0	391	86.3	114	82.6	P>0.05
Emphysema												
Male	Yes	4	7.7	2	7.1	2	16.7	8	8.7	14	12.7	
	No	48	92.3	26	92.9	10	83.3	84	91.3	96	87.3	P>0.05
Female	Yes	32	12.7	13	10.4	9	11.8	54	11.9	29	21.0	
	No	221	87.4	112	89.6	67	88.2	400	88.1	109	79.0	P>0.05
Metastasis												
Male	Yes	23	65.7	12	63.2	3	50.0	38	63.3	50	69.4	
	No	12	34.3	7	36.8	3	50.0	22	36.7	22	30.6	P>0.05
Female	Yes	93	66.4	42	66.7	29	59.2	164	65.1	55	67.9	
	No	47	33.6	21	33.3	20	40.8	88	34.9	26	32.1	P>0.05

Conditional logistic analyses of never smoker

When lung cancer cases (120) matched with non-respiratory system disease (120), the observed results were shown to fit the following equations:

Males: logit Pi=ai-1.330x_2+0.0481x_3
Females: logit Pi=ai-0.796x_2+0.032x_3+0.216x_5-0.548x_7

When lung cancer cases (120) were matched with non-respiratory cancer (120), results consistent with the following equations were obtained.

Males: logit Pi=ai+0.054x_3
Females: logit Pi=ai-0.663x_2+0.129x_5-0.217x_7

These results suggest that fresh vegetables (x_2) act as a protective factor against lung cancer, whereas contact with toxic substances (x_3) increases the risk of lung cancer. It is worth noting that in females, indoor air pollution (x_5) and situation of kitchen (x_7) are risk factors for lung cancer. However, the respiratory disease (x_1), ETS exposure (x_4) living conditions (x_6), and familial history of cancer (x_{10}), exerted no effect whatsoever on female lung cancer. The exclusion of cooking fuel (x_8) and participation in cooking (x_9) in regression equations might make it quite the same between the lung cancer cases and the matched controls. In the case of males, besides cigarette smoking, the major risk factors were related to occupational exposure.

A case--control study on never smoking females

The effects of spousal smoking on female lung cancer are illustrated in table 2 and table 3. The OR of ETS exposure is between 0.61--1.62 (P>0.05), showing that spousal smoking, measured either by daily cigarette consumption, or the duration of smoking, is not a risk factor for female lung cancer. Such a conclusion was reached both when the case control study was matched with non-tumor controls or controls involving non-respiratory tumor cases.

Table 2. Effects of (ETS) on never smoking females in 75 lung cancer cases and 128 controls (non-tumor deaths).

	Lung Cancer	Controls	Odd Ratio (95%CL)	x^2	P-Value
ETS refers to husband who smoke					
Yes	47	75			
No	28	53	1.19 (0.66--2.16)	0.33	>0.05
Total	75	128			
ETS refers to number cigarettes smoked per day					
0	28	53			
<20	13	34	0.72 /		
20-	30	35	1.62 (0.83-3.15)	4.03	>0.05
Total	71	122			
ETS refers to smoking years					
0	28	53			
<30	14	19	1.39 (0.61-3.16)	0.65	>0.05
30-	29	47	1.17 (0.60-2.29)	0.22	>0.05
Total	71	119			

ETS exposure and lung cancer cell type

The results of the comparison of lung cancer cell type between ETS and non-ETS exposure are shown in table 4.

The results indicated that no differences in cell types were observed between the exposed and non-exposed groups in both males and females, (x^2=1.76--3.78, P>0.05). In other words, exposure to ETS is not to be etiologically linked to an increase in epidermoid carcinoma of lung cancer .

Table 3. Effects of (ETS) on never smoking females in 75 lung cancer cases and 126 controls (tumor except lung cancer).

	Lung Cancer	Controls	Odd Ratio (95%CL)	x^2	P-Value
ETS refers to husband who smoke					
Yes	47	79	1.00 /		
No	28	47		0.00	>0.05
Total	75	126			
ETS refers to number of cigarettes smoked per day					
0	28	47			
<20	13	35	0.62 /		
20-	30	37	1.36 (0.73--2.56)	3.75	>0.05
Total	71	119			
ETS refers to smoking years					
0	28	47			
<30	14	18	1.13 (0.77--1.66)	0.47	>0.05
30-	29	49	0.99 /		
Total	71	114			

Table 4. Comparison of lung cancer cell type between ETS and non-ETS exposured groups in 192 of never smoking lung cancer deaths.

	ETS exposure(No. of Family Smoker)								Non-ETS exposure	
	1		2		3		Total			
	No.	%	No.	%	No.	%	No.	%	No.	%
Epidermoid ca.	6	50.0	2	40.0	4	66.8	12	53.0	9	30.0
Small cell ca.	0	0.0	1	20.0	0	0.0	1	4.0	0	0.0
M Adeno ca.	4	33.4	1	20.0	1	16.6	6	26.3	13	43.0
Large cell ca.	1	8.3	0	0.0	0	0.0	1	4.0	0	0.0
Others	1	8.3	1	20.0	1	16.6	3	13.7	8	26.7
Total	12		5		6		23		30	
Epidermoid ca.	15	22.4	6	24.0	3	16.7	24	21.8	5	17.2
Small cell ca..	4	6.0	4	16.0	1	5.5	9	8.1	3	10.3
F Adeno ca.	38	56.7	11	44.0	11	61.1	60	54.6	19	65.5
Large cell ca.	0	0.0	0	0.0	0	0.0	0	0.0	1	3.5

DISCUSSION

A number of investigators[1,2] concluded that an association did not exist between ETS exposure and lung cancer. However many other authors[3,4] emphasized the importance of ETS exposure as being causally linked to lung cancer.

In fact, any research pertaining to the effect of ETS on lung cancer is greatly restricted by a number of considerations, for example: (1) Only the "true" effects of ETS on never smokers can be evaluated, provided that never smoking subjects are available who are constantly, steadily exposed to ETS, and free from complications of other indoor pollutants and/or occupational exposures. However, such a condition is practically difficult if not impossible to achieve. (2) Questionnaires administered through the postal service make it difficult for some information to be obtained accurately. (3) Studies using only hospital based cases are confounded by selection bias. (4) The source of is ETS not likely to remain constant over an extended period of time. (5) In the case of spousal smoking, it is hard to eliminate whether there is "intentional avoidance" to ETS exposure, or whether "psychological conditioning" exists during ETS exposure. (6) Although probable carcinogens (BaP, DMNA) have been detected in sidestream tobacco smoke, and the concentration may be exceeding that present in mainstream tobacco smoke, they are undoubtedly greatly diluted when presented in the form of ETS, and are unlikely to reach the lower respiratory tract, like the mainstream; so that if lung cancer is induced by passive smoking, the major cancer type may be central epidermoid carcinoma and not peripheral adenocarcinoma. Apparently, when in order to confirm the effect of ETS on lung cancer, all of these factors must be carefully considered. Unfortunately, currently available data do not seem provide an adequate explanation on this subject.

Our studies showed that exposure to ETS had no associated with lung cancer, but it does not mean that ETS had no harmful to human health. There are more than one hundred chemical compositions that can be detected in sidestream tobacco smoke[5], a number of them being toxic substances.

REFERENCES

1. Lee PN, Chamberlain, Alderson MR, Relationship of passive smoking to risk of lung cancer and other smoking-associated diseases. Br. J. Cancer, 1986;54,97-105.
2. Williams AHW, Dai XD, Blot W, et al. Lung cancer among women in north-east China. Br. J. Cancer, 1990;62,982-987.
3. Wynder EL, Goodman MT, Smoking and lung cancer: some unresolved issues. Epidemiologic Reviews 1983;5:177-207.
4. Pershagen G,Hrubec Z, Svensson C, Passive smoking and lung cancer in Swedish women. American J. of Epidemiology. 1987;125(1):17-24.
5. Eatough DJ, Hansen LD, Lewis EA, The Chemical characterization of environmental tobacco smoke, Proceedings of the International Symposium at McGill University. 1989:P3-39.

EFFECTS OF RESTRICTIVE SMOKING POLICIES ON INDOOR AIR QUALITY AND SICK BUILDING SYNDROME: A STUDY OF 27 AIR-CONDITIONED OFFICES

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ABSTRACT

A field experiment investigated the effects of five restrictive or prohibitive smoking policie indoor air quality and sick building syndrome complaints in 27 air-conditioned office build Indoor air quality was measured in each building. No differences among policies were four numbers of smokers, average number of cigarettes smoked daily. No differences among were found for carbon monoxide, carbon dioxide, respirable particulates, relative humidity temperature or illumination levels. There were differences among policies in ultra-violet p mass and formaldehyde. There were differences in nicotine among the spatially restricted policies. ETS pollutant levels were highest where smoking was restricted to rooms with lo filtration. A questionnaire survey of workers measured sick building syndrome symptoms. Symptoms were marginally less prevalent for the restrictive smoking policies than the smo prohibited policy. Evidence that ETS is a cause of sick building syndrome complaints was found.

INTRODUCTION

Environmental tobacco smoke (ETS) is a source of *many* indoor air pollutants. Various spatially restrictive or prohibitive smoking policies can be implemented to lessen the impact of ETS pollutants on indoor air quality. Studies have shown that some restrictive smoking policies have little impact on indoor air quality (1,2). These studies, however, have not examined the effects of smoking policies on the sick building syndrome (SBS). Other work suggests that passive exposure to ETS increases SBS symptoms in nonsmokers (3,4,5), although smoking activity and SBS complaints are not associated (5,6). To investigate the effect of five smoking policies (prohibition and various forms of spatial restriction) on indoor air quality and on the SBS a field experiment was conducted.

METHODS

Smoking policies and office buildings

Five smoking policies were investigated: smoking prohibited (SP); smoking restricted to rooms with local electrostatic and sorbent air filtration units (RF); smoking restricted to areas with no local air treatment (RNT); smoking restricted to rooms ventilated by a separate exhaust ventilation system (RSV); smoking restricted to enclosed offices and open plan cubicle workstations (RWS). Twenty seven air-conditioned buildings with different smoking policies were studied. The buildings had either variable air volume (VAV) or constant air volume (CAV) ventilation systems. Seventeen organizations (insurance, finance, sales and marketing, etc.) occupied these offices. Fifteen of these were private companies occupying 25 of the 27 offices, 1 was a federal agency, and 1 a municipality.